

**MALATTIE DEL MIOCARDIO E DEL PERICARDIO 390**

COMPLICANZE DELLE PROCEDURE DI INTERVENTISTICA (*CARDIOLOGIA INTERVENTISTICA, CORONARICA E STRUTTURALE*)

ABLAZIONE TRANSCATETERE (*ARITMIE*)

PERICARDITI (*MALATTIE DEL MIOCARDIO E DEL PERICARDIO*)

IMAGING MIOCARDICO-PERICARDICO (*MALATTIE DEL MIOCARDIO E DEL PERICARDIO*)

FARMACI CARDIOVASCOLARI (*FARMACI CARDIOVASCOLARI E NUTRACEUTICI*)

**LATE PLEURO-PERICARDIAL EFFUSION AFTER ATRIAL FIBRILLATION  
RADIOFREQUENCY ABLATION**

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**Background:** post-cardiac injury syndrome (PCIS) is an inflammatory state involving pericardium, epicardium and myocardium causing a clinical picture in which epicardial and pericardial symptoms are prevalent. It appears mediated by autoimmune mechanisms and may appear as late post myocardial infarction pericarditis (Dressler's Syndrome) or as a post traumatic pericarditis in the case of spontaneous thoracic trauma or iatrogenic pericarditis. Apart from the acute setting, pericardial effusion can be a manifestation of PCIS after interventional procedures.

**Case summary:** a 57 years old hypertensive woman suffering from recurrent atrial fibrillation episodes underwent a technically difficult radio-frequency catheter ablation because of complex pulmonary veins anatomy and wide scar in the left atrial wall. During the procedure she developed cardiac tamponade and 410 ml of blood were drained by pericardiocentesis and re-infused without recurrent pericardial effusion during further in-hospital stay. She was discharged on apixaban 5 mg b.i.d. with Hb value of 10.2 g/dl. Two weeks later the patient was hospitalized for worsening cough, atypical chest pain, dyspnea and modest orthopnea. C-reactive protein levels were 8.7 mg/dl, Hb was 9.9 g/dl and platelet count 484.000/ml; blood cultures were negative. An urgent thoracic CT scan showed bilateral pleural effusion and ubiquitous pericardial effusion (2.5- 3 cm), without signs of active bleeding from the cardiac chambers into the pericardium. After stopping apixaban, the patient was given colchicine (1 mg/die). A total of 1200 ml of hematic pericardial fluid was drained from the pericardium over a 5-day period. Autoimmune blood tests were negative, as well as antibodies to pericardiotropic viruses. Pericardial fluid was negative for quantiferon and direct BK. On day 9, the drain was removed and steroidal treatment was started (prednisone 25 mg/die with scheduled tapering). Further echocardiographic exams were stable without pericardial effusion; a chest X-ray scan (at day 16) showed reversal of the water bottle shaped heart and of the pleural effusion.

**Discussion:** early myocardial infarct-associated pericarditis and Dressler's syndrome account for about 20% of cases of PCIS accompanied by symptoms of epicardial and pericardial origin. PCIS is quite common after cardiac surgery, but it may be also observed even after iatrogenic trauma occurring during cardiac interventions: PCI, pacemaker lead insertion, radiofrequency ablation and Swan-Ganz catheterization. Blood entering the pericardium is thought to play a pivotal etiological role in iatrogenic PCIS, with consequent huge inflammatory reaction in the mesothelial tissue resulting in clinical manifestations of pericarditis. In animal models of PCIS, systemic release of cardiac antigens and self-antigen specific responses has been hypothesized. In our case cardiac tamponade complicating the ablation procedure probably initiated the epicardial and pericardial inflammatory response. Even if based on few data, the patient was treated with colchicine first, avoiding aspirin because of the hemorrhagic pericardial fluid; glucocorticoids were then started when symptoms and signs of PCIS slowly resolved despite colchicine treatment. The pericardial fluid was hemorrhagic (Hb 5.9 g/dl) and treatment with apixaban, in the context of an inflammatory mesothelial response, could have caused this peculiar, hemorrhagic, pericardial reaction.